

Aortoduodenal fistula occurring after type II endoleak treatment with coil embolization of the aortic sac

Stéphane Elkouri, MD,^a Jean-Francois Blair, MD,^a Eric Thérèse, MD,^b Vincent L. Oliva, MD,^b Luc Bruneau, MD,^a and Gilles Soulez, MD,^b *Montréal, Québec, Canada*

A bifurcated stent graft device was successfully deployed to exclude an asymptomatic abdominal aortic aneurysm (AAA) with adequate proximal aortic neck morphology. At 6 months, a type II endoleak was successfully embolized through a proximal perigraft channel with metallic coils. The patient was seen with upper gastrointestinal bleeding and a pulsatile abdominal mass 11 months later. Surgical exploration revealed an aortoduodenal fistula in the vicinity of the previous embolization. We discuss the possible causes of this complication and review the literature on the subject. We conclude that aortoduodenal fistula can occur after endovascular AAA repair despite the absence of endoleak or AAA diameter increase on follow-up computed tomographic scan. (*J Vasc Surg* 2003;37:461-4.)

Endovascular repair (EVAR) of abdominal aortic aneurysm (AAA) is an emerging technique that could offer a less invasive alternative to conventional open surgery. Encouraging early and midterm results have been reported in spite of periprosthetic endoleaks. However, long-term benefits are still under evaluation. Absence of endograft incorporation,^{1,2} kinking of the endograft after aneurysm shrinkage,³ and persistence of pressurized AAA sacs without evidence of endoleaks^{4,5} question the durability and safety of this treatment with the currently available devices. We report the case of a patient with an aortoduodenal fistula 17 months after successful endovascular AAA repair.

CASE REPORT

A 75-year-old woman was referred to our center in December 1997 after the discovery of a 4.4-cm asymptomatic infrarenal AAA. The patient was followed with serial abdominal ultrasound scan until May 1999 when the aneurysm diameter had increased to 5.5 cm. Computed tomographic (CT) scan and angiogram both confirmed that the aneurysm was morphologically suitable for EVAR, having a 20 mm-long, tubular, noncalcified, and nonangulated proximal aortic neck. No signs of inflammatory aneurysm were found. The serum creatinine level was 118 mmol/L. After informed consent was obtained, a Talent bifurcated device (Medtronic, Sunrise, Fla) with a 24-mm main body and 12-mm iliac diameter was deployed via transverse common femoral arteriotomies in October 1999 without complications. The intraoperative completion angiogram confirmed adequate deployment of the device with the proximal bare portion of the stent at the level of ostia of the renal arteries. A CT scan done 48 hours later showed a

minor lumbar type II endoleak. The patient was discharged from the hospital on the fifth postoperative day.

The 1-month follow-up CT scan showed no increase in aneurysm diameter and spontaneous sealing of the type II endoleak. The 6-month follow-up CT scan in May 2000 showed stable aneurysm diameter but evidence of a new posterior endoleak. An angiogram was done that confirmed the presence of a type II lumbar endoleak. Embolization of the ascending branches of the right iliolumbar artery and superior gluteal arteries with 355- μ to 500- μ particles (Contour, Target Therapeutics, Fremont, Calif) failed to eradicate the endoleak. A lymphocele in the left groin (140 mL) was evacuated 1 week later, and culture of the lymph fluid showed 3+ presence of coagulase-negative *Staphylococcus*. Two weeks later, embolization of the left iliolumbar artery was attempted from the left common femoral artery without success because of inability to catheterize the left iliolumbar artery. It was decided to perform a catheterization of the aneurysmal sac via a left axillary artery approach. A 3F coaxial catheter was advanced between the prosthesis and the aortic wall to embolize the right posterolateral portion of the AAA sac with 0.018-in coils (Vortx, Target Therapeutics, Fremont, Calif) and thrombin (1000 units; Thrombostat, Parke-Davis, Scarborough, Ontario, Canada). A control CT scan 24 hours later showed small air bubbles around the coils with no residual endoleak (Fig 1). A follow-up CT scan done 3 months later, in September, showed no endoleak, no increase in aneurysm diameter, and no residual air around the coils.

The patient consulted shortly thereafter in another hospital for abdominal malaise, anorexia, and asthenia. Unexplained acute renal insufficiency was diagnosed, and the patient was referred to a nephrologist. A CT scan done in November revealed no signs of intraabdominal pathology and no abnormalities related to the endoprosthesis. Both kidneys were normally perfused, and AAA size was stable with no evidence of endoleak and no sign of endoprosthesis migration or infection. In March 2001, the patient consulted again for new onset of diarrhea. The patient also had nausea, vomiting, fever, and anorexia with a 30-lb weight loss over the preceding 6 months. The hemoglobin level, white cell count, and creatinine level were 83 g/dL, 2700/mm³, and 147 mmol/L,

From the Division of Vascular Surgery^a and the Department of Radiology,^b Centre Hospitalier de l'Université de Montréal (CHUM).

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Reprint requests: Stéphane Elkouri, MD, CHUM, 1560 Sherbrooke E, Montréal, Québec, Canada, H2L 4M1 (e-mail: elkouris@hotmail.com).

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Fig 1. CT scan 24 hours after coil embolization showed small air bubbles around coils with no residual endoleak.

respectively. Urinalysis results were normal. Twenty-four hours after admission to hospital, severe upper gastrointestinal bleeding with hematemesis and melena developed. An upper gastrointestinal endoscopy showed an adherent clot in the third portion of the duodenum. The patient was immediately transferred to our institution for a suspected aortoduodenal fistula.

On arrival, the patient was transferred to the intensive care unit in hemorrhagic shock. Physical examination revealed a pulsatile mass. An urgent CT scan showed massive retroperitoneal air around the endoprosthesis within the aneurysm and a retroperitoneal hematoma (Fig 2). The right kidney was atrophic without nephrogram. A plain abdominal radiograph showed no apparent change or metallic disruption. The patient was brought to the operating room and underwent an axillobifemoral bypass. At laparotomy, there was no hemoperitoneum. The retroperitoneal tissues anterior to the aneurysm were thickened and inflamed, and the AAA was pulsatile. The duodenum was adherent to the lateral portion of the aneurysm. After clamping of the suprarenal aorta and both common iliac arteries, the aneurysmal sac was opened. The endoprosthesis appeared intact with no disruption of its body or its attachment to the left iliac branch. However, it had migrated proximally and covered the origin of the right renal artery but had not perforated the aortic wall. On its right side below the aortic neck, we found the cavity that had been embolized previously. This cavity was stained with bile, contained multiple coils, some of which were embedded in the aortic wall, and communicated with the third portion of the duodenum through a 1-cm fistula. No bleeding was noted at the level of the cavity. The endoprosthesis was not incorporated to the surrounding aortic wall, with possible poor proximal sealing, and was easily removed in its proximal portion at the level of the aortoduodenal fistula (Fig 3). An infrarenal aortic stump was created and covered with greater

omentum. The duodenal fistula was closed, and the region was drained. Unfortunately, the patient died 12 hours later in refractory septic shock.

Culture results of sample taken from within the aneurysm sac revealed mixed aerobic and anaerobic flora (coagulase-negative *Staphylococcus*, nonenterococcal group D *Streptococcus*, *Prevotella melaninogenica*, *Fusobacterium nucleatum*, *Bacteroides uniformis*, and *Fragilis* and *Peptostreptococcus micros*). Ex vivo macroscopic examination of the stent graft revealed no structural fractures, but a small tear was found in the fabric, probably of iatrogenic origin during the operation.

DISCUSSION

Secondary aortoenteric fistula is a rare complication of open AAA repair. The incidence rate of this complication after EVAR is unknown. To our knowledge, five other cases of aortoenteric fistula occurring 5 to 22 months after endovascular AAA repair have been reported. In the absence of an anastomosis suture line, three distinct situations in which this complication was reported may help to understand the pathophysiology of fistula formation after EVAR.

In two instances, aortoenteric fistula formation was reported in patients with a preexisting inflammatory process around the aneurysm shown on CT scan. The first case was reported in 1998 by Norgren, Jernby, and Engellau.⁶ The complication occurred 17 months after a successful endovascular AAA procedure with a first generation Stentor device (MinTec, Ltd, Freeport, The Bahamas). In the second case, the aortoenteric fistula occurred 7 months after apparent successful AAA exclusion with an AneuRx device (Medtronic AVE, Santa Rosa, Calif) with no apparent mechanical stent failure, distortion, or migration.⁷ In both cases, the etiology of the periaortic inflammatory mass was uncertain, but the resolution of the inflammatory process before the complication occurrence lends support to the diagnosis of an inflammatory rather than a mycotic aneurysm. However, the pathogenic mechanism responsible for the fistula in these cases remains unknown, and stenting of a primarily infected AAA cannot be excluded.

Poor fixation and poor sealing were also identified as possible causes of fistula formation.^{8,9} Hausegger et al⁸ reported an aortoduodenal fistula occurring 20 months after EVAR. This patient had marked angulation of the proximal neck that resulted into migration and kinking of the device against the anterior wall of the aneurysm. Subsequent erosion of the aortic wall by the stent graft was proposed as a potential cause of fistula formation. The upper section of the bifurcated Vanguard device (Boston Scientific, Watertown, Mass) was found to be disconnected as a result of suture disruption. d'Othee et al⁹ reported a similar case with proximal aortic neck enlargement resulting in fixation failure. Device migration and fracture and repressurization of the aneurysmal sac led to erosion of the aortic wall.

Finally, primary stent graft infection has also been proposed as the possible cause of aortoenteric fistula formation 5 months after EVAR in a patient admitted to

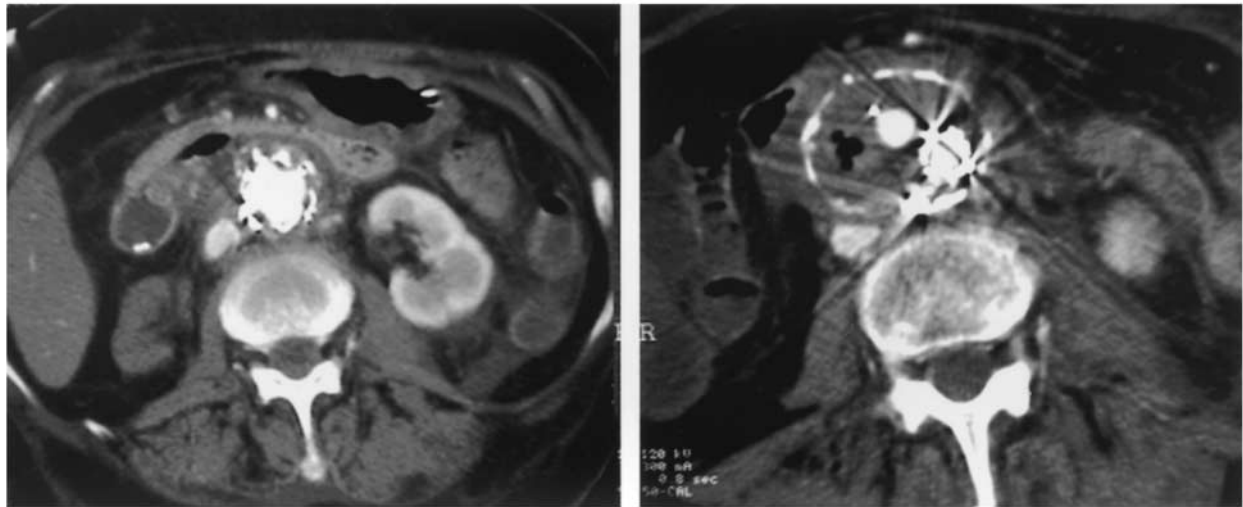


Fig 2. CT scan at 17 months showed massive retroperitoneal air around endoprosthesis within aneurysm and retroperitoneal hematoma.

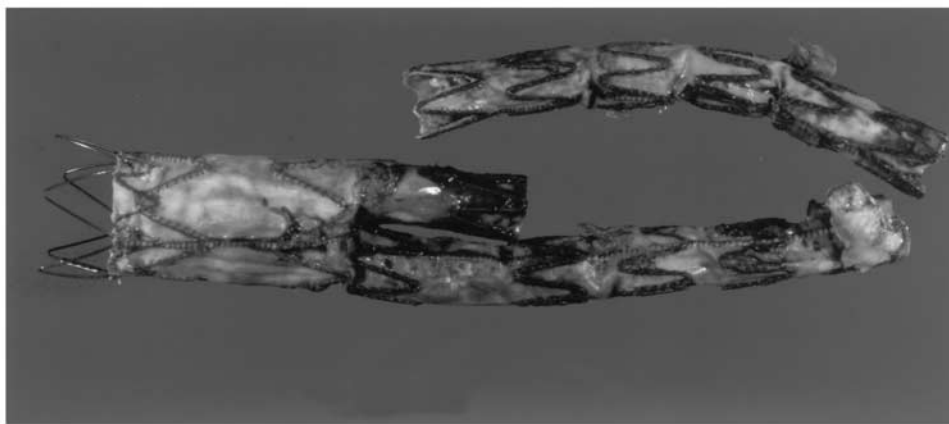


Fig 3. Talent endoprosthesis after surgical explant.

hospital with pneumonia and no radiologic evidence of endoleak, increase in aneurysm size, stent migration, or device failure.¹⁰ According to the authors, an infected stent graft may have led to septic aortitis and subsequent fistula formation.

In this case, there were no clinical or radiologic signs of a primary aortoduodenal fistula before EVAR. Despite the apparent successful AAA exclusion from blood flow and the absence of detectable endoleak or increase in AAA diameter on follow-up CT scan performed 4 months previously, the patient was seen 17 months after EVAR with an aortoduodenal fistula that resulted in death. Of interest, the fistula was found to be in communication with the AAA sac at the level of the cavity embolized 9 months before treatment of a type II endoleak. Coils embedded in the aortic wall and stained with bile were found in proximity to the fistula orifice. These findings suggest a relation between the treat-

ment of the type II endoleak and the subsequent aortoduodenal fistula.

The significance of type II endoleak remains uncertain. Recent reports indicate that type II endoleaks are not associated with a higher risk of AAA rupture or conversion and that treatment should therefore only be performed in case of increase in aneurysm size.¹¹ In this case, the fistula formation may be a complication of the type II endoleak treatment. The disappearance of air bubbles after the last embolization and the absence of radiologic signs suggesting infection on the September and November CT scans indicate that a virulent infectious process primarily responsible for this complication is less probable. However, a low-grade infection of the stent graft and aortic wall cannot be excluded. The treatment of an infected lymphocele in the left groin 1 week before the two endoleak embolization procedures, one of which was done through the left groin,

may have resulted in infectious contamination during the procedure. Coagulase-negative *Staphylococcus* was cultured from both the lymphocele and the aneurysm sac. Also, an injury to the aortic wall or prosthesis during the embolization procedure cannot be excluded, although manipulation with a 3F microcatheter and embolization with soft 0.018-in coil is unlikely to be traumatic. The presence of metallic coils embedded into the aortic wall in the region of the fistula suggests that chronic and repetitive aortic wall trauma may have occurred. Pulsatility of the stent graft device or endotension in the sac may have exacerbated the wall injury from the metallic coils. The combination of aortic wall injury and septic aortitis may explain fistulization into the duodenum.

The endoleak treatment done through a periprosthetic track may have resulted in endotension from an occult, incomplete seal of the proximal anchoring system.¹²⁻¹⁶ Successful endoleak embolization through direct translumbar puncture of the aneurysm sac was reported by several authors, thereby avoiding catheterization of the proximal attachment zone.¹⁷ The proximal migration of the device indicates poor fixation despite the ideal aortic neck morphology in this patient and despite the bare stent portion of the Talent graft. Poor fixation may be inherent to inadequate fixation with this technology or the results of infectious aortitis, neck enlargement, or periprosthetic passage during treatment of type II endoleak.

We conclude that aortoduodenal fistulization and aneurysm rupture can occur after EVAR despite the absence of detectable endoleak or increase in AAA diameter on follow-up CT scan. The findings in this case suggest that aortoduodenal fistulization could be a complication of treatment of a type II endoleak. Metallic coils within the AAA sac and embedded within the aortic wall may have produced chronic repetitive aortic wall injury from pulsation of the stent graft device or endotension and may have led to fistulization and aneurysm rupture. Clinical and radiologic follow-up every 6 months after EVAR did not permit early detection and prevention of this fatal complication.

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